

Combination of very low LDL and normal systolic blood pressure attenuate coronary artery disease

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New data published in the March 31, 2009, issue of the *Journal of the American College of Cardiology* show that patients with coronary artery disease (CAD) who achieve very low levels of low-density lipoprotein (LDL) cholesterol along with normal systolic blood pressure have the slowest progression of CAD. The results suggest that patients with CAD should be treated to the most stringent target levels so that they can achieve optimal results from their lipid lowering and antihypertensive therapies.

"This paper has a simple but important message regarding dual targets for prevention of coronary artery disease," says Adnan K. Chhatriwalla, M.D., interventional cardiology fellow at the Cleveland Clinic, Cleveland, Ohio, and lead author of the study. "It is the first study to demonstrate that normal blood pressure and very low LDL cholesterol in combination are associated with attenuation of the progression of coronary disease in humans. Even though patients may have reasonable control of blood pressure and cholesterol, getting them to optimal treatment goals is best in terms of slowing plaque progression."

Dr. Chhatriwalla and his colleagues studied changes in atheroma burden as monitored by intravascular ultrasound (IVUS) in 3,437 patients with CAD. The patients were stratified based on LDL cholesterol greater or less than 70 mg/dL and systolic blood pressure greater or less than 120 mmHg. Four different measures of plaque progression were studied: Percent atheroma volume, total atheroma volume, percent of patients with significant plaque progression, and percent of patients with significant plaque regression.

For all of those variables, there was less progression of disease in patients who achieved LDL-cholesterol? 70 mg/dL and systolic blood

pressure ? 120 mmHg in combination.

"The finding that patients who were able to get their LDL below 70 mg/dL and their systolic blood pressure below 120 mmHg had the slowest progression of heart disease supports the growing concept that more than one risk factor is driving the disease," says Stephen J. Nicholls, assistant professor of molecular medicine at the Cleveland Clinic, and co-author of the study. "The thought really needs to be that the greatest bang for your buck in terms of preventing heart disease is going to be by trying to have aggressive control of all the risk factors."

Chhatriwalla et al. "provide an interesting commentary on the need for more aggressive treatment guidelines for 'pre-hypertension,' i.e, blood pressure of 120 to 139/80 to 89 mm Hg in patients with established coronary artery disease," comment Jonathan Tobis, M.D. and Alice Perlowski, M.D., from the David Geffen School of Medicine at UCLA, Los Angeles, California, in an accompanying editorial.

However, they caution that IVUS, although a robust clinical and research tool, is subject to limitations. "Although we may use plaque progression/regression on IVUS to deduce that we are producing positive results for our patients, the true determination of the impact of our therapy depends on clinical and mortality end points, which can only be obtained from large-scale randomized clinical trials."

Dr. Chhatriwalla maintains his study sends an important message. "This study suggests that when it comes to cholesterol and blood pressure targets, 'close' is not good enough. We need to stress that patients with the best risk factor control have the best clinical results. This is the take-home



message."

Source: American College of Cardiology (<u>news</u>: web)

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